Th. Ed.

CHRONIC INTESTINAL STASIS.

T H E S I S

submitted for the

M.D. EDINBURGH 1925

by

HAROLD BALFOUR DYKES,

M.B., Ch.B., Ed. 1917.

Late House Physician,
Royal Infirmary,
Edinburgh.

CHRONIC INTESTINAL STASIS.

CHRONIC INTESTINAL STASIS.

I. INTRODUCTION.

In the present thesis the writer proposes to discuss the present-day views on the subject of Chronic Intestinal Stasis.

The subject of Chronic Intestinal Stasis is a wide and interesting one, and one that has engaged the attention of many of the foremost medical and surgical men in this and other countries. And well might it be, for the condition is one which is not peculiar to any race or clime. It occurs among all classes, rich and poor alike, in both sexes and at all

ages. Every medical man must meet with patients who present the features of it. It is of frequent occurrence, varying, of course, in degree.

In many cases Chronic Intestinal Stasis may exist without demonstrable cause, or there may be some definite pathological condition (e.g., visceroptosis) at the bottom of it. Its effects are far-reaching. Metchnikoff[1.] stated in 1907 that "The accumulation of waste matter retained in the large intestine for considerable periods becomes a nidus for microbes which produce fermentations and putrefaction harmful to the organism." He was of opinion that the products of intestinal putrefaction stimulate the activity of the phagocytes and so encourage their encroachment on the higher tissues: and he concludes that "Intestinal Putrefaction

shortens life."

Since the work of Arbuthnot Lane, Chronic Intestinal Stasis has been the subject of a large amount of medical literature. In many cases it is very difficult to distinguish the condition from ordinary constipation, but, in the light of Lane's work, many of those cases of ordinary obstinate constipation may be looked upon as cases of Chronic Intestinal Stasis. Lane has done more than anyone else in connection with the condition - so much so that Victor Pauchet of Paris suggests that Chronic Intestinal Stasis should be termed "Arbuthnot Lane's Disease."

Chronic Intestinal Stasis is, as the name implies, a condition of stasis in the intestinal contents. But there is some confusion in the

use of the term. It is more often used to apply to the clinical picture which is considered to result from a state of chronic stasis.

Using the term in the strict sense we understand that there is an unusually great period of time during which the contents of the alimentary tract are retained within that tract. Lane - using the expression to describe the clinical picture - defines the condition of Chronic Intestinal Stasis as one in which there is delay in the passage of the contents of the intestine at one or more points; this delay leading to bacterial infection of the contents with increased decomposition and putrefaction of them - the resulting toxins being absorbed into the circulation in quantities too great to be neutralised by the ordinary agencies of the body (e.g. the

liver) and thus causing a general poisoning of the body - "Auto Intoxication" - which manifests itself by a degeneration of every tissue in the body and by certain definite clinical characters.

The starting point is thus a delay in the passage of the contents of the alimentary tract. Now the rate of passage of these contents varies very much in different individuals, and moreover in the same individual at different times. Some persons suffer from malaise, headache, pain, and a furred tongue, if they fail to obtain a daily evacuation of the bowels: while others are perfectly healthy and happy with an evacuation regularly once a week or even at longer intervals, and may show none of the features of auto-intoxication, or, as it is termed, alimentary toxaemia.

2.

Roux gave the term coecal constipation to the condition where there is stagnation of faeces in the proximal part of the colon. Stagnation here, he says, is of more serious consequence than stagnation in the distal end of the colon: for in the coecum the faeces are in a liquid state and bacterial activity is very intense. Here the residues of digestion may be broken up into toxic substances which when absorbed give rise to uncomfortable and perhaps serious symptoms. A person with stagnation of faeces in the rectum or distal colon may go for several days without experiencing any discomfort: whereas where there is retardation of faeces in the small intestine or coecum for even a short time, the subject complains of abdominal fulness, with nausea, headache,

and diminished appetite, i.e. symptoms of digestive intoxication.

We see then that the mere retention of faeces (or coprostasis) is not necessarily followed by auto-intoxication. Hence it is obvious that the conditions of Intestinal Stasis and alimentary Toxaemia are not identical, and that one is not the necessary result of the other.

Here it may be fitting to describe Lane's toxic type, the victim of auto-intoxication. The toxins attack every organ and every tissue, and seem to attack especially those organs and tissues that are normally engaged in excreting toxins, e.g., the skin, kidneys, and the liver (which neutralises toxins). The main effect of the toxins is to cause degeneration of

tissues and loss of fat. The patient is more often a woman than a man. She becomes thin, nervous, melancholic, depressed, neurasthenic, and obstinate constipation is the rule, the constipation often progressing in degree. The skin becomes thin, inelastic, sallow, wrinkled, and pigmented, especially over those areas exposed to pressure. Pigmentation begins in the eyelids, spreads over the face, and neck, axillae, inner aspects of thighs and over the vertebral spines. The sweat glands secrete excessively, the sweat being often offensive. The breasts undergo changes beginning with an induration in the upper and outer quadrant of the left breast and spreading throughout both breasts, passing into a state of cystic degeneration. The muscles become flabby and lose

tone so that some of the joints (e.g. the knees especially in children) are capable of hyper-extension. There is a loss of subcutaneous fat, the breasts droop, the abdomen protrudes and the buttocks become less prominent. The circulation is depressed and the extremities, especially the feet, become cold, livid, and often have impaired sensation. (They may resemble the extremities in Raynaud's disease.)

II. ANATOMY, COMPARATIVE ANATOMY AND PHYSIOLOGY OF THE ALIMENTARY CANAL.

Before proceeding to the anatomy of the alimentary canal, it is of value to recall the general anatomy of the abdominal cavity. It is roofed in by the tent-like diaphragm. Its floor comprises the structures which clothe the

inner surface of the bony pelvis - principally the levator ani, the coccygeus and the obturator fascia. The posterior wall is formed by the lumbar and sacral vertebrae with the psoas major and quadratus lumborum muscles. The anterior wall is composed of muscles and fascia entirely: the external and internal obliques and the transversalis muscle, the recti with their sheaths, and the pyramidales, and the strong transversalis fascia. The side walls are formed by continuation of the oblique muscles and the transversalis. The whole cavity is lined with the parietal layer of peritoneum.

In dealing with the question of Chronic Intestinal Stasis, it is well to bear these walls in mind as any abnormality in them (either congenital or acquired) may produce some corresponding

abnormality in the position of the contained organs; and this may, in turn, have some delaying effect on the passage of food and waste products along the alimentary canal.

In passing to the important matter of the attachments of the bowel, we recall the distribution of the peritoneum in its parietal and visceral layers and the consequent formation of the greater and lesser sacs of the peritoneal cavity.

The bowel is attached to the walls of the abdominal cavity by and receives support from mesenteries which are formed by the visceral peritoneum as the latter is reflected from the posterior walls to cover the viscera. There are several mesenteries - the mesentery proper, the mesentery of the appendix, the transverse mesocolon, the sigmoid mesocolon and sometimes the mesocolon of

the ascending and the descending colons.

The mesentery proper is the broad fan-shaped fold of peritoneum connecting the convolutions of the jejunum and ileum with the posterior wall of the abdomen. Its root is narrow - about 6 inches long - and is directed obliquely from the duodeno-jejunal flexure at the left side of the second lumbar vertebra to the right sacro-iliac articulation. The intestinal border is nearly 22 feet long. This mesentery is short above but lengthens rapidly to about 8 inches and is thrown into innumerable folds. It suspends the small intestine with its blood-vessels, lymphatics and nerves.

The mesentery of the appendix is attached to the back of the lower end of the mesentery proper close to the ileocoecal junction and passes behind the terminal part of the ileum to cover the

appendix.

The transverse mesocolon is a broad fold suspending the transverse colon to the posterior wall of the abdomen. It is continuous with the two posterior layers of the great omentum which after separating to surround the transverse colon join behind it and are continued to the vertebral column where they diverge in front of the anterior border of the pancreas.

The sigmoid mesocolon is a fold of peritoneum which retains the sigmoid colon in connection with the pelvic wall. Its line of attachment begins on the medial side of the left psoas major and runs upwards and backwards to the apex of its V-shaped curve which is near the point of division of the left common iliac artery: thence it bends sharply downwards and ends in the medial plane at the

level of the 3rd sacral vertebra.

Although, as a rule, the peritoneum covers only the front sides of the ascending and the descending colon, sometimes these portions of the large intestine are invested by peritoneum on all sides and attached to the posterior abdominal wall by a mesentery. According to Treves the descending colon possesses a mesentery more frequently than the ascending.

In addition to receiving support from the mesentery and mesocolons, the bowel gains additional support from folds of peritoneum which have received names corresponding to the organs they "connect". The hepatogastric ligament is that portion of the lesser omentum extending between the liver and the stomach, while the hepatoduodenal ligament is that portion between the liver and the duodenum. The

splenic flexure receives additional support from the phrenicocolic ligament, a fold of peritoneum which passes from it to the diaphragm opposite the 10th and 11th ribs.

The position of the stomach varies with the posture, with the amount of gastric contents, and with the condition of the intestine on which it rests. In the erect posture the organ is somewhat J-shaped: the part above the cardiac orifice is usually distended with gas: the pylorus descends to the level of the 2nd lumbar vertebra and the most dependent part of the stomach is at the level of the umbilicus.

The small intestine is a convoluted tube about 22 feet long, wider at its commencement than at its termination, which is contained in the central and lower parts of the abdominal cavity

and is surrounded above and at the sides by the large intestine. In front it is in relation with the great omentum and the abdominal parietes, a portion of it descends into the pelvis and lies in front of the rectum. The small intestine consists of a short portion, the duodenum (which is devoid of a mesentery), and a long greatly coiled part (which is contained in the mesentery) which in its proximal two-fifths is called the jejunum and in its distal three-fifths the ileum.

The duodenum is the shortest, widest and most fixed part of the small intestine. It has no mesentery and is only partially covered with peritoneum. It is curved on itself, its end being but a short way from its starting point: the curve is filled in by the head and neck of the pancreas. It ends opposite the 2nd lumbar

vertebra in the jejunum where it takes an abrupt forward bend and forms the duodeno jejunal flexure. The last short portion of duodenum is held in position by a small strand of combined muscular and fibrous tissue which has been called the musculus suspensorius duodeni and which arises from the right crus of the diaphragm close to the right margin of the oesophagus and is attached to the posterior aspect of the upper part of the duodeno-jejunal flexure.

The jejunum for the most part occupies the umbilical and left iliac regions of the abdomen while the ileum lies chiefly in the umbilical, hypogastric, right iliac and pelvic regions. The ileum ends by opening into the medial side of the coecum in the right iliac fossa.

The large intestine, which is about 5 feet

long, is divided into the coecum, the ascending colon, the transverse colon, the descending colon, the sigmoid colon, the rectum, and the anal canal. Its calibre is largest at its commencement and gradually diminishes until it reaches the rectum where again it is greatly dilated just above the anal canal.

The coecum lies in the right iliac fossa on the iliacus and psoas muscles. It is about 2½ inches long and the same in width. As a rule it is in contact with the anterior abdominal wall but the great omentum and some coils of small intestine may intervene. In about 95% of cases the coecum is entirely enveloped in peritoneum; while in the other 5% the covering by peritoneum is not complete - the upper part of the posterior surface being uncovered and attached to the iliac fascia by

connective tissue. The coecum enjoys a considerable amount of movement; it may become herniated down the right inguinal canal and has even been found in an inquinal hernia on the left side. The vermiform appendix is a long narrow blind tube communicating with the coecum, which starts from what was originally the apex of the coecum. It has its own mesentery.

The ileo coecal valve which separates the ileum from the coecum requires some mention. Presenting a slit-like appearance as seen from the coecum, the opening runs at right angles to the long axis of the bowel. The edges of the valve are thickened and form lips which are reinforced by muscular fibres. This valve allows of the passage of contents of small intestine through to the coecum, but nothing from the latter can re-

enter the ileum except in rare instances and under great pressure.

The large intestine passes on from the coecum as the ascending colon which, as has been detailed above, rarely has a mesocolon: at the under surface of the right lobe of the liver the ascending colon merges into the transverse colon at the hepatic flexure. The transverse colon is contained in its mesocolon and crossing the abdomen in front of the middle portion of the duodenum to the spleen it ends at the splenic flexure by taking a sudden bend downwards and becoming the descending colon. This portion of the large intestine runs down through the left side of the abdomen and on a level with the crest of the ileum, at the outer border of the left psoas, it becomes the sigmoid colon. The descending colon is, as a rule, gen-

erally uncovered with peritoneum on its posterior aspect, but occasionally has a mesocolon.

The sigmoid colon normally lies within the pelvis. It is about eight inches long and is looped, the loop consisting of three parts: the first part descending in contact with the left pelvic wall, the second crossing the pelvic cavity (between the rectum and bladder in the male, and between the rectum and uterus in the female), sometimes as far as the right pelvic wall, and the third arching back to reach the middle-line at the level of the third sacral vertebra. Here it becomes continuous with the rectum. The sigmoid colon has a mesocolon which is longer at the centre than at either end, and so the centre portion of this piece of the colon has a wide range of movement.

The rectum seems to be wrongly named in the

human as ,the terminal part of the colon, instead of being straight as its name implies, it presents two flexures. It begins at the level of the 3rd sacral vertebra, is about 5 inches long, lies in the sacrococcygeal curve, runs downwards for about 3 inches and bends upon itself, passing backwards and downwards for another 2 inches and ends in the anal canal. The upper two thirds of the rectum are covered in front and on the sides by peritoneum, but the lower third has only a slight covering in front.

The anal canal is the terminal portion of the bowel: it is about 1 inch long and is surrounded by the internal sphincter, the levator ani and at its absolute termination by the external sphincter.

The stomach wall is composed of four coats: the serous, muscular, areolar and mucous. The

muscular coat consists of three layers of unstriped muscle - longitudinal, circular and oblique, the first named being to the outside. The mucous membrane is thrown into folds and depressions and contains innumerable glands. Between the four coats of the wall run the blood-vessels, lymphatics and nerves.

The wall of the small intestine consists of the same four coats as that of the stomach. The muscular coat consists of two layers, an external longitudinal layer and an internal circular layer. The mucous membrane is formed into the valvulae conniventes, and contains the intestinal villi, intestinal glands or crypts of Lieberkühn, Brunner's glands (in the duodenum only) and Peyer's patches (mainly in the ileum).

The wall of the large intestine too has the

same four coats. The muscular coat differs from that of the small intestine in that the longitudinal layer does not form a continuous layer over the surface. In the coecum and colon the fibres are grouped into 3 bands, which being shorter than the other coats of the wall produce the sacculation of the bowel. The fibres become scattered again at the sigmoid colon and reaching the rectum form a continuous covering. The mucous membrane of the large intestine contains glands and solitary lymphoid nodules.

The mucous membrane of the rectum is as a rule thrown into three transverse folds which have been called Houston's valves, the purpose of which, according to Patterson, is to support the weight of faecal matter.

The nerve supply of the gastro-intestinal

tract is a double one: some branches coming from the cerebro spinal and some from the sympathetic division of the nervous system. The former are excito-motor in action, the latter inhibitory to intestinal movements. So far as the ileo coecal junction the gastro-intestinal tract is supplied by the vagi; beyond by the pelvic nerves which have arisen from the sacral cord. The stomach and small intestine receive their sympathetic supply from the splanchmic nerves which arise in the lower dorsal and upper lumbar portions of the cord (their cell station being in the solar plexus): the colon and rectum receive theirs from fibres which arise in the lumbar region of the cord and have their cell station in the inferior mesenteric plexus.

The food contents are mixed and passed along

the intestinal tube by movements of the stomach and intestines. Those of the stomach are more particularly of a complex churning character: those of the small intestine are typically peristaltic; in addition there is a pendulum movement. The peristaltic movement can take place upwards or downwards, though normally it travels downwards. A certain amount of retroperistalsis takes place normally in the ascending colon. These movements are effected without consciousness, but they may be stimulated or inhibited in many ways. The usual stimulus is the presence of food material in the intestine and especially of indigestible food. The mere taking of food or liquid may stimulate peristalsis, e.g. breakfast or even just a glass of water on rising which produce a call to defaecation. Chemically by means of drugs one can stimulate or

inhibit peristalsis. Chemical substances, e.g. the internal secretions or hormones derived from the ductless glands have different actions on the nerves of the gastro intestinal tract: those from the thyroid gland,[3.] the spleen[4.] and the intestinal mucosa[5.] stimulate peristalsis through their action on Auerbach's plexus, while the internal secretion of the suprarenals[6.] has been demonstrated to stimulate the sympathetic ganglia. Temperature influences the movements of the bowel: hot food stimulates, cold quietens, but cold enemata are more effective than warm which are mainly sedative. Emotion plays a part: excitement or fear may stimulate peristalsis so much as to produce diarrhoea.

Food is digested, absorbed and assimilated. In the mouth, by mastication it is minced and mixed with the saliva, which contains ptyalin. This ferment aids in the digestion of starch, liquefying it

and converting it partially into dextrine and maltose. This action continues in the stomach until it is stopped by the acidity of the gastric juice. In the stomach, the pepsin and rennin of the gastric secretion take on the work of digestion: the proteins being split into peptones and a slight amount of fat splitting taking place. The food passes on into the duodenum, where it is mixed with the bile and the ferments from the pancreas. Here the peptones are resolved into their constituent amino-acids, starch is split into maltose and fats are split up into glycerol and fatty acids. Passing on from the duodenum the food comes under the action of the succus entericus which contains the activating agent Enterokinase which stimulates the trypsin from the pancreas in breaking up peptones.

Many of the bacteria that grow in the intestinal

contents produce enzymes which act similarly to the pancreatic juice. But certain actions are entirely due to the putefactive organisms. They act on carbohydrates, producing carbonic acid, hydrogen and butyric acid. They produce lower acids (valeric and butyric) from fats. From proteins they release indol, skatol and phenol. They act on amino acids to produce amines which if absorbed and not excreted by the kidneys may cause harmful effects.

The movements of the stomach and intestines mix the food contents with the gastric and intestinal juices, and so allow of the more intimate action of the ferments on the food.

The stomach takes little, if any part in the absorption of food. The great absorbing area is the mucous membrane of the small intestine, which

if spread out measures approximately 45 square yards. Absorption takes place gradually as the digested food passes along the alimentary tract and so the digested food gradually diminishes in quantity as it passes down the bowel and the faeces contain the undigested or indigestible residue. The products of digestion have almost disappeared by the time the contents of the bowel reach the ileo coecal valve. The colon takes little part in the process of digestion but does play a very important role in the process of absorption, especially of water, the coecum being the part of the large intestine where most of the absorption of food takes place. We know that some foods can be digested in the lower part of the colon - e.g. nutrient enemata.

The time taken for the complete emptying of the stomach is, on an average, about three hours,

but of course it varies with the size of the meal, its digestibility, and the general state of body and mind of the individual. The more fluid the food the more rapidly does it leave the stomach. The average time taken by food to reach the coecum is four hours from ingestion. In six hours the food reaches the hepatic flexure, in nine hours the splenic flexure, in eleven the iliac colon and in twelve it reaches the pelvic colon. These figures represent the average times and are Hurst's.[7.]

On arrival in the coecum the contents contain about 90% of water, the remaining part being composed of a small amount of the unabsorbed products of digestion of proteins, fats and carbohydrates, some superfluous digestive juices, as well as indigestible food material. Here and along the colon the excess of water and the previously unabsorbed

contents are absorbed; so that when the sigmoid colon is reached the contents are of the consistency of normal faeces, i.e. 75% water, and the rest made up of digestive juices, residue which has not been or could not be absorbed, undigested food material, bacteria, epithelial debris and a small amount of mucus. The amount, consistence, shape, colour and reaction of the faeces varies im health and disease, The healthy individual should have at least one copious evacuation of faeces in twenty-four hours. The average daily discharge is 4 - 6 ozs. The motion is of a firm or doughy consistence, cylindrical in shape, light or dark brown in colour, of offensive odour and ordinarily alkaline.

The arrival of faeces in the rectum constitutes the natural stimulus for defaecation, which is a reflex act capable of initiation or resistance

by voluntary action. The consequence of the stimulus is an orderly series of movements: (1) contraction of the rectum, the abdominal muscles and the diaphragm; (2) contraction of the levator ani and of the sphincters. The voluntary response to the stimulus consists of contraction of the abdominal muscles with a passive condition of the sphincters. If a person wishes to resist the excitant, there is a passive condition of the abdominal muscles with a contraction of the sphincters. When the stimulus is weak, the desire lacking, and the voluntary effort insufficient to commence the act, the call to defaecation soon passes off and may not return until the next regular period arrives, perhaps twenty-four hours later: if this occurs the faeces lose part of their water content, and get harder and so more difficult to expel.

This is one of the causes of constipation.

Inefficient defaecation may thus be due to habitual disregard of the call to defaecation, depending upon laziness, inability to obey the call immediately, ignorance, uninviting lavatories or unpleasant accompaniments of the act, e.g. pain or haemorrhage from the bowel.

Weakness of the defaecation reflex leads to similar results, and may be present congenitally or be required through organic or functional nervous disease, or by reason of reflex influences, or a deficiency in the bulk of faeces.

It is interesting to compare the anatomical structure of the intestinal tract in the vertebrate series. In the lowest group - fish - the large intestine is the least developed part of the tract,

being little wider in calibre than the small bowel. The first development of a coecum is found in some reptiles where it is represented by a lateral outgrowth from the large intestine. In birds there are a pair of more or less developed coeca at the point of junction between large and small intestines: in large ground birds such as ostriches and others the coeca are largest. In the mammal group the large intestine becomes more and more developed as we ascend the scale, being most developed in man.

Gegenbaur[8] writes that "the hind-gut is largest in mammalia, where it forms the large intestine, and is distinguished, as such, from the hind-gut or small intestine. Owing to its great length it is arranged in coils, so that the terminal portion only has the straight course taken by the hind-gut of other vertebrates".

What is the function of the large intestine? It has very little, if any, digestive power, but it helps in the absorption of water. It acts as a storage chamber for the refuse of digestion. Its mucous membrane secretes mucus which lubricates the walls for the passage of its contents.

Metchnikoff[9] was of opinion that "the large intestine which is useful to mammals the food of which consists of rough bulky vegetable matter, and which require a large reservoir for the waste of the process of digestion, is certainly useless in the case of man," citing as evidence the case of a woman who lived for 37 years although the large intestine was atrophied and inactive.

It has been pointed out by various observers that birds and lower vertebrates are longer lived than mammals and we know that the large intestine

is less developed in the former than in the latter: the deduction is that the higher development and state of the large bowel has some direct bearing on the length of life.

III. THE ETIOLOGY AND DIFFERENTIAL DIAGNOSIS OF THE VARIOUS FORMS OF CHRONIC INTESTINAL STASIS.

Before discussing the Etiology, Diagnosis and Treatment of Chronic Intestinal Stasis, it were well to consider the production of auto-intoxication or alimentary toxaemia -- i.e. the poisoning of the body which occurs in some persons as a result of Chronic Intestinal Stasis.

Leaving aside toxins which are ingested in food, we have the two factors to cause the poisoning:

I. Delay in passage of intestinal contents.
II. Bacteria.

We shall discuss the cause of the delay in the section on Etiology.

The bacteria of the alimentary tract may be roughly classified as:

(i) Coli group.

(ii) Various streptococci.

(iii) Certain anaerobes.

These bacteria have all adapted themselves to their habitat.

In the intestinal contents we find a medium rich in various types of foodstuffs, with plenty of moisture and warmth. Free oxygen is largely absent, except in certain diarrhoeal conditions. In the colon there is no free oxygen, and pure anaerobes can flourish. The normal inhabitants of the alimentary canal are facultative anaerobes and many of them have adapted themselves to live in the presence of bile and bile salts.

The streptococci found are of much less virulence than the streptococci pyogenes. They belong to various types, the most common being streptococcus salivarius and s. faecalis. Of the bacilli, B. Coli Communis is the commonest. B. Lactis aerogenes and B. Acidi Lactici are also common and other types sometimes occur, e.g. the Gaertner group, etc.

Of the pure anaerobes the commonest is the B. Enteritidis Sporogenes or B. aerogenes capsulatus, but other forms may occur. Other occasional inhabitants are B. pyocyaneus and B. proteus vulgaris.

Considering the various divisions of the alimentary tract, we find that in the mouth strepococci predominate very largely. The contents of the stomach and duodenum are perfectly sterile, the

bacteria swallowed in food and from the mouth being destroyed by the Hydrochloric acid in the gastric secretion.

Passing down the jejunum and ilium we find bacteria again, and they become more numerous as we get further down the ilium. The contents of the greater part of the small gut and of the large gut are alkaline and so form a good medium for bacteria as opposed to the acid contents of stomach, duodenum and upper part of jejunum. It is in the coecum and colon however that bacterial growth is most prolific. Members of the Coli group generally predominate in the large bowel. In the lower parts of the colon we find members of the strict anaerobe group - B. aerogenes capsulatus, etc.

About 90% of the masses of bacteria excreted in the faeces are dead but the fact remains that the

bowel, and more especially the large bowel, is teeming with living bacteria.

The delay in the passage of the contents of the bowel in Chronic Intestinal Stasis allows of the formation of toxins - either those produced in the tract as a result of decomposition, fermentation and putrefaction of ingested food or bacterial toxins. The latter, bacterial toxins, are practically negligible; the bacteria that are normally in the alimentary tract producing no soluble exotoxin, and apart from B. Coli no important endotoxin (these endotoxins of B. Coli are not believed to produce any appreciable general toxaemia).

Hence we are left with toxins formed by splitting up of food - both by chemical action and by bacteria.

In the normal course of food metabolism various end-products result. Fats are split up into lactic,

diacetic, oxybutyric acid, acetone, etc. These are absorbed and mostly rapidly altered and combined so as to render them harmless. If they are present in large quantities a certain amount is excreted in the urine. Proteins are split up into amino acids and these are absorbed and carried to the liver and reconstructed if the liver is not diseased. (Mutch).

10.

In cases where the liver is the seat of widespread degenerative change, e.g. in acute yellow atrophy, it is possible that auto-intoxication may occur, since the organ is incapable of dealing with the toxic products carried to it from the bowel.

In the bowel the food contents are not merely subjected to the action of the gastro intestinal juices but are also subjected to the action of

the swarms of bacteria present. Many of them, especially the strict anaerobes in the lower colon, are capable of splitting protein bodies to a stage considerably beyond that which is the end result of proper digestion and it is suggested that these final products of the fermentation, putrefaction and decomposition of proteins by the bacteria of the colon are responsible for toxic symptoms. Experimentally it has been found that the end products so far discovered - indol, skatol, phenol and hydrogen sulphide, etc. - are not markedly toxic, so that it would appear that there must be other final end products, as yet undiscovered, some of which are too toxic to be neutralised by the liver, etc. In a recent article, Shaw[11.] shows that the formation of formaldehyde by bacteria when grown on suitable media has a significance

of very considerable importance: and points out that this property may, in the case of certain organisms normally present in the intestinal canal, have a direct influence on orderly metabolism. He points out that it is evident from his experiments that the amount of formaldehyde present in the alimentary canal will entirely depend on the types of coliform organisms existing in it - for example, if organisms similar to Group IV are mainly present, the quantity of formaldehude produced will be minute, but if they are of Group III type a relatively large amount of this toxic substance may require to be dealt with. Shaw says that it must now be recognised that formaldehyde may be normally present in the alimentary canal, in degrees varying with the conditions obtaining, the kind

of food taken and the nature of the bacterial flora; and the possible effects of this compound on cellular metabolism deserve consideration in view of its extreme toxicity and immense range of chemical interaction.

The long delay in the passage of the bowel contents in cases of Chronic Intestinal Stasis allows the proteins to be split up into these final toxic end products. The stasis in the lower colon causes damming back of the more fluid contents of the coecum and lower ilium which probably leads to increased bacterial infection of the upper part of the bowel with increased putrefaction and fermentation here. It is to this ascending bacterial infection of the lower part of the small intestine as a result of ilial stasis, with the resulting putrefaction of the contents, that Lane ascribed 12.

the symptoms and results of auto-intoxication, the toxins, according to this theory, being largely absorbed from the decomposing contents of the ilium and not so much the colon.

Chronic constipation leads to a state of catarrhal inflammation in the bowel mucosa: this may even pass on to ulceration and perhaps perforation of the inner coats of the bowel. Through this damaged wall of the bowel it is very probable that toxic substances can pass in larger quantities than can be dealt with completely by the liver, thyroid, etc. These harmful products enter and for a certain period remain in the general circulation and produce effects on the different organs of the body. And so "alimentary toxaemia" occurs.

13.

Metchnikoff considered that the symptoms

were produced in such a way, but rather emphasized the possibility of organisms or poisons, or both, passing directly through to the blood stream owing to a breach in the surface of the mucosa. Microscopically the bowel wall in cases of well marked alimentary toxaemia shows pathological changes in its substance.

14. Gant inclines to agree that the incidence of the toxic symptoms may be the result of some lesion of the intestinal mucosa, but he considers that no one has been able to give a satisfactory explanation of it.

We can thus understand how Chronic Intestinal Stasis can lead to auto-intoxication.

In reviewing the Etiology of Chronic Intestinal Stasis there are certain predisposing causes

to be mentioned.

Sex. Females are much more liable to the condition than males. This may be explained by the fact that women are generally confined to the house more and lead a more sedentary life than men: they take less exercise. Pregnancy favours constipation naturally by pressure on the intestines by the gravid uterus. Ovarian and uterine tumours add to the frequency with which women suffer from chronic constipation.

Age. Elderly people and babies show the condition more than older children, young people and the middle-aged. The aged lack vitality, their glands are inactive and their intestinal musculature is atrophied. In infants there are many anatomic causes of constipation: e.g. the great length of the intestines and mesentery tend

to promote twists and angulation: the muscular bands of the bowel are yet but poorly developed, hence there is deficient peristalsis. The natural position of the infant is on its back and this is against the proper use of its abdominal muscles. Bad training accounts for a great deal of constipation in babies.

Occupation and Environment: Generally speaking, people who work hard and out of doors suffer less from chronic constipation than those who do not do manual labour outside. Those who go in for games, etc. are less apt to be constipated than those who follow the same work but do not take active recreation.

Impairment of the Abdominal muscles. Pregnancy causes overstretching of muscles and separation of the fibres: so leading to weakness. Obesity produces

fatty degeneration of the muscles and atony through the constant pressure of a heavy and fat abdomen.

Atony of the Intestinal Walls follows prolonged distension or is the result of operations on the gut.

Determining causes are legion.

Dietetic Errors are responsible for constipation in many cases, e.g., the taking of too little fluid, of too little fat, too much meat or too few vegetables.

Irregular hours of defaecation. The healthy individual more generally than not has a regular time for defaecation, but some people ignore the call to stool and so from day to day constipation becomes more and more marked. The most common time for the daily action of the bowels is after

breakfast, which meal stimulates peristalsis.

Chemical and Medicinal Causes. Astringents such as copper, alum, iron and lime salts may be ingested in food or drinking water in quantities sufficient to lessen the evacuations from the bowel. It must not be forgotten that some people take quantities of patent medicines (e.g. those containing bismuth) over a long period without their doctor's knowledge, and these will certainly cause constipation.

General diseases, e.g., pleurisy, pneumonia, and tuberculosis which interfere with the proper movements of the diaphragm may be cited as causes of constipation. Diseases of the liver and gall bladder producing deficiency in the supply of bile tend to produce constipation. In fevers, the supply of water in the body finds other outlets than in the faeces (e.g. by the skin), and so the

percentage of water in the faeces is lessened and this leads to constipation. Inflammatory diseases of the bowel wall or mucosa such as colitis, sigmoiditis and proctitis by leading to a thickening of the muscular coats of the intestine, diminish its power of contraction - hence constipation may result. Mucous colitis commonly produces obstinate constipation.

Now we come to the large group of Mechanical Causes of Chronic Intestinal Stasis.

Of these by far the most important and common cause is Visceroptosis.

We have seen that the large and small intestines are suspended by and gain support from mesenteries and other accessory bonds of tissue which are considered to be normal. But on examining the abdomen in many cases of Chronic Intestinal Stasis

one meets with various accessory layers of the peritoneum forming the mesentery of the bowel, or definite bands (or pseudo-peritoneal membranes with kinks or angulations of the bowel where these bands meet it.)

The commonest of these are found:

(i) Spreading obliquely downwards and inwards from the parietal peritoneum on the outer side of the colon over the coecum and ascending colon as a veil-like sheet of membrane which has been called "Jackson's Membrane".

(ii) A band passing from the parietal peritoneum in the region of the sacro-iliac joint to the ilium within a few inches of the ilio-coecal valve - the Lane's ilial band.

15.

(iii) Lane has given the name "First and Last Kink" to a twist in the bowel at the junction

of the descending and pelvic colons caused by another band of "adhesions".

(iv) Another band (Payr's Membrane) which pulls the splenic flexure upwards emphasising the already existing acute bend there.

These bands are quite definite. The membranes are not firmly fixed to the underlying peritoneal surface of the gut, but are separated by an oedematous subserous layer, or may be quite free over most of their extent so that a finger can be inserted between veil and bowel.

There is much discussion about the origin of these bands of membrane. There are three theories: that they are:

I. Inflammatory.
II. Congenital, or
III. Acquired evolutionary supports.

I. The Inflammatory Theory which Jackson put forward in his original description ascribes the membranes to be inflammatory adhesions, resulting from appendicitis or colitis. The term "membranous pericolitis" has been applied to the condition by those who support this theory: similarly perisigmoiditis for similar bands in the region of the sigmoid.

Against the Inflammatory Theory are the following facts:

(a) The appendix may be absolutely normal and yet there may be extensive membranes reaching over the coecum and ascending colon.

(b) The adhesions run down along the outer aspect of the colon - and the vessels run in parallel lines downwards and inwards through the membrane - an arrangement which would not be

expected if vascularisation occurred in inflammatory adhesions.

(c) There is no thickening along the lymphatics which run along the course of the vessels to the bowel. Thickening would be expected if the adhesions were inflammatory in nature. Moreover, the thickened bands or membranes are not most marked along the course of lymphatics and vessels - but are on the outer aspect of the bowel.

II. With regard to the Congenital Theory, there is no doubt that in some cases these bands and membranes are present at birth or in the full term foetus.

One theory of this formation is embryological, viz. that the coecum and lower end of the ilium in their descent from beneath the liver burrow behind

the posterior parietal peritoneum of the abdominal wall, parts of which remain spread out over the bowel as "Jackson's Membranes". This theory is probably incorrect.

Another theory is that they result from foetal peritonitis - a lesion which, it is known, may occur in utero.

16.

The third theory - on the congenital side - is that the bands and membranes result from a process of physiological evolution: they are due to the process of fixation of the mesenteries (peritoneal zygosis as it has been termed) proceeding too far. In the early foetus there is at first a condition of the bowel in which the whole intestinal tract is supported by a single fan-shaped mesentery attached posteriorly at one spot only - i.e. where the vessels enter -

this being described as complete zygosis of the mesentery i.e. complete nonfixation. As development proceeds the mesentery becomes fixed at certain spots, viz.:

(i) In the postgastric area, the mesentery containing the pancreas and duodenum becoming applied to the posterior abdominal wall and being merged in it.

(ii) In the mesocolic area, the transverse mesocolon becoming adherent to the posterior abdominal wall.

At a later stage the ascending and descending colon become similarly fixed to the abdominal wall by a process of peritoneal zygosis. This process occurs as follows:

The outer leaf of the mesocolon becomes applied to the posterior parietal peritoneum in

the loin, a proliferation of the subserous tissue occurs in the angle between parietal peritoneum and base of mesentery, with the result that the "angle" is carried further and further outwards till finally the colon is lying on the abdominal wall and the "angle" of peritoneal reflexion lies on its outer aspect. This process is somewhat alien to an inflammatory or healing process. This process of peritoneal or mesenteric fixation in the foetus is very variable in the time at, and the extent to, which it occurs. It is particularly variable in extent in the region of the iliac colon and iliocoecal valve. In both of these places the bowel may not be completely fixed down at birth and moreover in 50% of cases at birth the process has over-reached the usual extent in the region of the iliocoecal valve with

the result that the lower few inches of ilium with the iliocoecal valve region and coecum are completely fixed down to the posterior abdominal wall.

According to Keith this is the origin of Lane's ilial band, the lower end of the ilium becoming mobilised later on in life, by drawing away from the posterior abdominal wall, the original adhesion remaining on Lane's band and causing Lane's Kink

Similarly, the right extremity of the great omentum may possibly in course of development become adherent to the hepatic flexure and outer aspect of the ascending colon in the neighbourhood, and later in foetal life when the coecum etc. descend from beneath the liver, this "adhesion" becomes drawn down with the colon and constitutes

Jackson's Membrane.

III. The third theory, that the bands are acquired evolutionary structures, is Lane's. According to this theory, the fault lies in prolonged standing or sitting in the erect posture. The contents of the ascending colon fall into the coecum which becomes distended and sags into the pelvis downwards and inwards. This direction of coecal dropping may be considered the resultant of a parallelogram of forces, the outer and inner limbs of which become "crystallised lines of strain or resistance", these lines of strain being marked first as a thickening in the peritoneum supporting the bowel, later as definite bands and finally as membranes of peritoneum. The outer limb develops as thickenings, bands and membranes passing downwards over the coecum and ascending

colon from the parietal peritoneum on its outer side, i.e. "Jackson's Membrane".

Jackson's Membrane and the ilial band bend to hold up the coecum and so counteract the downward drop of the viscus.

As against the inflammatory origin of the ilial band Lane points to the fact that it originates in the base of the mesentery and gradually extends to the bowel: and moreover it is only present on the under aspect of the mesentery where it will have an effective upward pull.

The appendix may be caught either in the outer limb of the parallelogram when it will be drawn upwards and outwards, or in the inner limb when it will be drawn up and inwards. In either case the appendix is liable to become kinked, its distal part distending and appendicitis

resulting.

The next stage in the process of evolution is that the bands contract and though they thus give better support to the coecum, yet they cause kinking and angulation of the bowel especially of the lower part of the ilium.

The ilial kink is thus produced. This ilial obstruction is increased if the appendix is caught up in the inner line of the parallelogram since in such a case the appendix runs up behind the end of the ilium and is fixed to the iliam nesentery, or to the ilial band, thus narrowing the superjacent ilium where they cross one another.

The ilium proximal to the kink becomes distended and the loaded coils fall into the pelvis, thus increasing the kink and still more obstructing the ilial effluent leading to ilial stasis.

The distended ilium drags on its mesentery and so kinks the duodenojejunal flexure - perhaps also causing partial torsion of the beginning of the jejunum with the result that the duodenal effluent is obstructed and the duodenum dilates. This leads to spasm of the pylorus which in turn leads to dilatation of the stomach which tends to prolapse, and so leads to further crystallised lines of strain in the shape of adhesion between the pylorus and gall bladder or liver.

And so the process goes on interminably. The loaded transverse colon drags downwards, and so bands and membranes form in the region of hepatic and splenic flexures to support it. These bands contract and kink the bowel at the flexures.

Similarly membranes and bands form on the

outer side of the mesosigmoid to support the loaded sigmoid. These may contract, drawing the two ends of the loop close together causing kinks and predisposing to volvulus. The bands may form irregularly and so kink the centre of the sigmoid loop. The last kink of the series is where the sigmoid passes over the pelvic brim. This produces the "First and Last Kink" of Lane. According to Lane this last kink is a very important physiological one and is formed very early, even before the child begins to walk, as its function is to prevent regurgitation of faeces upwards into the descending colon.

Finally in many cases of stasis the rectum is elongated and bent to form an "S" so that attempts at defaecation cam only empty one limb of the "S" and expand the rectum laterally.

This is really a condition of Dyschezia or difficulty in defaecation.

In Lane's opinion the kinking of the bowel by these bands especially in the sigmoid and at the splenic flexure, may lead to the formation of diverticula which when inflamed produce the condition Diverticulitis as described by Dr. W. J. Mayo.

Such is Lane's theory of a drainage system completely at fault, the large bowel forming a cesspool which cannot be properly emptied and infection and stagnation extending up all along the gastro-intestinal tract. In support of his theory he demonstrates kinks and angulations at operation and his co-worker Jordan demonstrates delay in the passage of opaque meals, the delay occurring at certain spots where kinks or adhesions can be demonstrated and which are sometimes

tender. Lane again demonstrates hypertrophy and dilatation of the bowel above the kinks, pointing to obstruction, though in the later stages the bowel is often shrunken, thin and flaccid.

Against Lane's mechanical theory of stasis are the following points:

1. In many cases of tubercular etc. peritonitis where the whole bowel is a mass of kinks and angulations no auto-intoxication occurs, and obstruction even is not present.

2. Many cases showing marked kinks have no auto-intoxication during life.

3. The giving of an opaque meal is not a satisfactory test of the normal action of the bowel.

4. Many of the thin membranes are too fragile to cause obstruction to the lumen of the bowel.

It is more probable that the primary fault

lies in the muscle of the bowel wall, this being incapable of proper peristalsis.

Sections of these bowels show pathological changes in bowel wall and ganglia. Perhaps the membranes so interfere with distension of the bowel and peristalsis and have some slight action in this way.

In the normal body the viscera do not drag on the mesentery, but are supported by the intra-abdominal pressure. It is possible that one and the same cause may be responsible for visceroptosis, from weakening of the abdominal muscles, and stasis, from weakening of the bowel wall. Visceroptosis is very frequently, almost invariably, associated with intestinal stasis.

17.

By some the ptosis of the abdominal organs is regarded as the primary factor in the produc-

18.
tion of Chronic Intestinal Stasis, but Hurst is probably correct in his view that more frequently the two conditions are only associated together because both are independently produced by the abnormal condition of the abdominal and pelvic muscles. Others think that the membranes are the cause of the ptosis.

19.
It has been shown by Smith and others that there are two varieties of skeletal change which have been called the virginal and the maternal types.

In the virginal type, the patient is thin and poorly developed: the thorax is narrow and shallow; the upper abdomen is narrow and the waist long, the shoulders droop, the lower abdomen protrudes and droops; as a rule the condition is acquired in childhood, though in some cases

there is an hereditary factor. The changes are usually called "The Ptosis Habitus".

The maternal type does not show skeletal changes so that the thorax is unaltered. The lower abdomen bags and protrudes owing to the weakness of the abdominal muscles due to repeated pregnancies: the lumbar curve is lost and the shoulders are round.

Many of the people who show these changes do not suffer any inconvenience.

20. Walton suggests that it is only in a patient who, having membrane formation, later develops ptosis that we get symptoms produced.

21. Conran suggests using the term "Hypotomic Diathesis" instead of the "Ptosus Habitus" of Walton: he defines it as a particular condition or habit of body predisposing to a loss of tone

in the musculature of the alimentary canal and commonly associated with a low position of the abdominal viscera.

Passing from Visceroptosis to the other mechanical causes of Chronic Intestinal Stasis, these are many.

The more common of these causes are tumours of the bowel or abdominal tumours of any sort, faecal impaction, diseases of the rectum and anus (Haemorrhoids, etc.), adhesions, volvulus, hernia, intestinal calculi, internal parasites, diverticula, strictures (following dysenteric ulcer or colitis, etc.) foreign bodies and congenital malformations.

DIAGNOSIS.

Chronic Intestinal Stasis affects all the systems of the body, and so the symptoms vary

accordingly.

As can be readily understood, digestive symptoms are as a rule the first complained of or elicited: furred tongue, bad taste in the mouth, foul breath, nausea, thirst, poor appetite, flatulence, vomiting, abdominal tenderness, colic, perhaps occasional attacks of diarrhoea and the passage of mucus with the motions.

Nervous symptoms met with in Chronic Intestinal Stasis vary from slight dull headache or neuralgia, etc. to loss of memory, neurasthenia and hysteria - with flashes in front of the eyes, giddiness and insomnia, as other in-between nervous manifestations. Convulsions are commonly seen as a result of chronic constipation in children.

The urinary system frequently suffers and patients complain of frequency of micturition,

dark coloured urine and increasing deposits. Albumen is sometimes found in the urine.

The circulatory system suffers to the extent of palpitation and coldness of the extremities, this latter sometimes going on to a point where it resembles Raynaud's disease.

Arterio sclerosis, according to Evans[22], is an inflammatory condition evoked by bacterial intoxication from chronic Intestinal Stasis.

The Blood Picture alters in cases of Chronic Intestinal Stasis, as shown by White Robertson[23]. He shows that there is generally a decrease in the total number of red cells, a colour index slightly below normal (0.9 generally but frequently 0.86 to 0.84), and a decrease in the total number of white cells with a slight relative lymphocytosis. As the intestinal stasis becomes more marked, the

anaemia progresses relatively.

The skin shares in the general pathological change. Arbuthnot Lane[24.] notes as the most marked change in the skin the staining about areas exposed to friction, e.g. neck, spine, axillae, groins, and genitals. Sweat is increased and is of a bad odour. The temperature of the skin is subnormal, and an abrupt decrease in heat is noticeable in the extremities: the nose and ears are blue and cold: chilblains are frequent.

The eyesight as shown by Clarke[25.] in Lane's book (p.247) becomes affected as a result of intestinal toxaemia; the hardening of the lens comes on prematurely and so accommodation becomes weaker and weaker.

Mutch[26.] from his observations on 200 cases of multiple arthritis in which gout, venereal disease

and tuberculosis could be excluded, reports that stagnation was as common in the small gut as in the colon: it was present in 90 per cent. of his cases.

27. Chapple considers that Intestinal Stasis is an important etiological factor in the production of gynecological diseases.

Chronic mastitis is an early and constant sign.

Cancer as a result of Chronic Intestinal Stasis: 28. Jordan supports the theory of Lane that cancer is, to a large extent, secondary to toxaemia of intestinal stasis.

Generally speaking, the sufferer from Chronic Intestinal Stasis ages prematurely.

29. Lane ascribes all manner of diseases to Chronic Intestinal Stasis (the visceroptosis

type): he regards it as the direct cause of gall stones, gastric and duodenal ulcers; as the forego-er of tubercular disease, rheumatoid arthritis, diseases of the thyroid gland, etc., etc., and many others.

Auto-Intoxication may predispose its victims to any disease, but on the other hand, almost any disease may occur in persons in whom no intestinal stasis can be demonstrated. 30. Hurst points out that in the absence of any source of infection the possibility of an intestinal origin cannot be over-looked.

The history of the case is very important in diagnosing the cause of Chronic Intestinal Stasis. There is the time over which the patient has been constipated; whether constipation is increasing; the colour and consistence of the motions; and any

additional constituent such as blood or mucus. One may find a history of an old chronic metallic poisoning (e.g. lead), of dysentery, typhoid, or colitis (leading to stricture), of appendicitis or peritonitis (leading to adhesions). The occupation of the individual and the amount of exercise taken may be useful points.

The manual examination of the abdomen may reveal the cause of the constipation, e.g. tumour formation; it will show the general strength of the abdominal walls, points of tenderness.

Perhaps the most important information in suspected cases of Chronic Intestinal Stasis is gathered from a series of X-ray examinations at intervals after the patient has taken a meal intimately mixed with two to four ounces of barium sulphate. Hurst was the pioneer in X-ray

work in intestinal stasis, and his figures (given before) are considered as standard. He regards X-ray examination as indispensible to success in treatment.[31.]

It should be noted that in diagnosing stasis more stress is laid upon the time of disappearance of the meal from any particular part of the bowel than upon the time of its arrival there.

But, as pointed out before, the results obtained with barium cannot represent exactly the progress of ordinary intestinal contents along the alimentary tract. In this Hurst agrees, pointing out in his article, "The Sins and Sorrows of the Colon", that it is not necessarily pathological for the transverse colon to be looped into the pelvis, for the lower border of the stomach to be well below the umbilicus, or for the coecum to

drop into the pelvis in the upright position - facts often forgotten in reading radiographs of opaque meals.

Examination of the faeces is of importance. Here we may gain valuable assistance - nay, even discover the cause of the obstruction, e.g. intestinal calculi, gall stones or parasites.

In cases where no definite cause can be discovered by the foregoing methods the anal region and the rectum should be examined. The latter can be done by the finger or with the aid of the proctoscope.

Of course with some causes of Chronic Intestinal Stasis nothing definite can be found with any of the above methods: then recourse may have to be had to an exploratory laporatomy.

Differential Diagnosis. This is far from

simple, apart from chronic constipation resulting from rectal and anal diseases, foreign bodies and congenital malformations.

With tumours of the bowel or of some other organ in the abdominal cavity or with faecal impaction, diagnosis is much more simple, as a rule, than in cases of volvulus, adhesions, visceroptosis, diverticula, and strictures. Of course X-rays would be the deciding factor in diagnosing the last two. The three causes giving very similar symptoms are adhesions, volvulus and visceroptosis. Perhaps with adhesions there will be a history of old appendicitis or peritonitis.

With visceroptosis, as a rule, nothing objective can be discovered by the examination of the abdomen, except that generally the abdominal muscles are flabby and when the patient stands

erect the abdomen droops. Lane says there are tender spots in the abdomen which are points where there are kinks in the bowel.

IV. TREATMENT.

There are three groups of symptoms to be considered in treatment:

(i) Gastro Intestinal - flatulence, nausea, vomiting, pain, tenderness, etc.

(ii) Stasis - constipation, distension, attacks of incomplete, perhaps complete, obstruction.

(iii) Auto intoxication.

The superficial treatment is symptomatic but the bedrock of treatment is to seek the cause of the chronic stasis and eradicate that cause.

Treatment resolves itself into two kinds:

(a) Non-operative.

(b) Operative.

(a) Non-operative treatment. There are two objects in view:

1. To prevent the formation of toxic substances
2. To ensure a total evacuation of the ascending colon.

Causes predisposing to Chronic Intestinal Stasis must be counteracted: people must be encouraged to pay attention to the necessity of having regular daily evacuations; those whose work is of a sedentary nature should be instructed and encouraged to take as much open air exercise and recreation as possible; those whose work tends to favour the ptosis habitus must be educated to resist it by all means in their power and given sufficient means of resting and exercising.

Constipation in the vast majority of cases is acquired, and in many instances the habit is

formed during the early years of life. Consequently a most important prophylactic measure is the patient education of children in the subject of bowel hygiene: they should be taught to attempt to open the bowels each morning after breakfast and never to neglect to obey the call to defaecation, as this would result in the desire passing away. Clean and inviting lavatories should be the aim in all dwellings. The best form of water closet seat is one so planned that the person using it can assume a position as near as possible to the primitive squatting position as seen in the less civilised peoples.

Diet. This is very important, not only in the treatment of Chronic Intestinal Stasis but also in its prevention.

One frequently finds dietetic errors in

patients suffering from constipation, and when these errors are corrected, the action of the bowels becomes regular and more frequent. The general error is the taking of too much meat. Nitrogenous substances, especially when they have not been acted on sufficiently by gastric juice, are dangerous. Albumens in solution (especially in lightly cooked eggs) are to be avoided. Milk is not often well borne. In certain patients with great degrees of fermentation and formation of gas in the colon, leguminous foods such as peas, beans and lentils are to be forbidden. The diet should be primarily vegetarian.

The cellulose content of food is all important. The proportion of cellulose varies considerably in different vegetables, but those found most valuable in the prevention or treatment

of constipation are whole meal, outmeal, spinach, cabbage, asparagus, onions, tomatoes, parsnips, watercress, celery and lettuce. The potato as ordinarily cooked and eaten is of little value: but, if baked and eaten with the skin, it is of considerable value. Fruit, fresh or cooked, should be eaten at most, if not all, meals. In addition to cellulose it contains an important quantity of sugar and organic acids which are valuable because of their chemical properties in stimulating the motor activity of the bowel. Prunes, raisins, dried figs, dates (all cooked or uncooked) and fresh fruit such as apples, pears, oranges, pineapples, melons, strawberries, plums, peaches, currants, grapes, cherries, raspberries and gooseberries are useful in counteracting constipation and some of them are

available at any season.

Fats increase the motor activity of the intestines and can be given in the form of butter, salad oil, cream, olive oil, etc.

Proteins are required for metabolism and provided they are not excessive in quantity may be allowed in the diet.[32.] Mutch points out that the protein content of the food has to be substantially reduced in cases where intestinal toxaemia shows itself, until improvement occurs.

Fluids are of importance. Plenty of water, preferably cold, should be drunk. Cold water stimulates, while warm water reduces peristalsis. Tea is not advisable in combatting constipation, the tannin being the harmful constituent. Wines stimulate peristalsis slightly, red wines less so than others.[33.] Metchnikoff advocated the use of sour milk because of its anti-putrescent

action.

34.

Hutchison gives the following type of diet:

Morning Meal: Tea or coffee, prepared with water. Bread, butter, jam or cooked fruit.

Mid-day Meal. Thick soup with rice or macaroni. Grilled or roast meat, or fish. Well cooked potatoes or fresh vegetables. Pudding (Semolina, tapioca or rice). Fruit, cooked or raw.

Four p.m. Tea, bread with butter or cooked fruit.

Evening Meal. The same as at lunch time, but without meat or fish.

Mechanical. Abdominal support may be provided by a Curtis (or other) Belt. Great care must be taken with the measuring and fitting of these belts, as if the pressure is applied at the wrong point, more harm than good may result.

General Exercises and Massage. There are many different varieties of exercises which are

helpful in combatting chronic constipation. A few simple but very useful exercises are the following:-

I. Lie on back: arms by the side, legs outstretched. Raise one leg to perpendicular, then lower. Repeat with other leg. Raise both legs together to perpendicular, then lower.

II. Lie on back: legs outstretched: hands stretched above head. Raise trunk at hips to vertical sitting position, and slowly resume position.

III. Stand erect: hands on hips: chin drawn in. Incline trunk forward from hips to an angle of forty-five degrees from the upright. Then bend right forward from hips as far as possible, back arched. Resume second position. Resume original position.

IV. Trunk Rolling. Stand as for III. Bend forward at the hips, then roll trunk round to left, then to back, then to right and so on. Keep feet firmly on the ground.

Under the term exercises are included, of course, gymnastics and all games. Climbing, rowing and riding are of most value, because they develop the abdominal muscles and cause frequent alteration in the intra-abdominal pressure.

This form of treatment is of great value in cases of visceroptosis. Walton[20.] points out that any course of exercises must include adequate rest so that the overworked muscles may recover: the aid of gravity must be called in to return the viscera to their normal position (thus many of the exercises should be performed

with the patient in the prone position): the skeletal muscles and body shape must be improved, special attention therefore being paid to improving the lumbar curve and widening the lower thorax: the body fat must be increased (this to be aided by increasing the amount of rest); and the stasis must be overcome.

Massage. This may be either by hand or by mechanical vibration. Where neither of these is available, there is one method of proved merit which may be carried out by the patient himself - that is rolling a six-pound shot over the abdomen along the course of the colon.

Lubricants. The most important element in treatment is to secure thorough evacuation of the coecum. Of lubricants, medicated paraffin takes first place. This is now made up with malt and other constituents into appetising form by many

manufacturers, e.g. in Nujol, Virolax and Crystolax, etc., all of which I have found of value in the constipation of infants and children.

Medicinal. Saline purgatives: The most generally useful of this group are Magnesium and Sodium salts. By some they are supposed to produce their action by Osmosis[35], but Hurst[36] believes the salts are absorbed and act through the vessels directly on the colon as stimulants to movement and secretion. They are safe, pretty reliable and rapid in their action. They are especially useful in constipation associated with hepatic disorder, gout, or excessive uric acid. Sodium sulphate and Magnesium Sulphate are combined in most mineral waters, e.g. Carlsbad, Marienbad, Hunyadi Janos, Seidlitz, Rubinat, Apenta and Kissingen waters.

Aloes: In the intestine aloes cause an

increase in the rate of the flow of bile and probably the amount secreted. It produces little influence in the small intestine, but the muscular coat of the colon is powerfully stimulated. Aloes therefore are slow in their action but can be used over long periods, although as it acts chiefly on the lower bowel the habitual use of it may lead to haemorrhoids.

Cascara Sagrada is a simple laxative and aperient. It is very useful as gradually increased doses are not required.

Rhubarb. - more used as a stomachic than a purgative - acts, it is generally believed, by exaggerating very actively intestinal peristalsis. Purgation is followed by constipation which is ascribed to the rheo-tannic acid.

Senna - a very safe, useful purgative for

cases of simple constipation. It stimulates the muscular coat of the intestine especially of the colon.

Castor Oil is perhaps the safest and most reliable drug we have for constipation. It does not gripe. It is rather unpleasant to the taste, but the writer usually orders it to be taken in hot milk or hot Bovril.

Calomel. The action of calomel is chiefly on the duodenum and upper part of the jejunum. The contents of the duodenum are hurried along before there is time for the bile to be reabsorbed or altered. There is some increase in the secretion from the intestinal walls. As the action of calomel is chiefly on the upper part of the intestine, its action is greatly assisted by giving a saline purge a few hours after it, for this will act more on the lower part of the bowel.

Belladonna and Atropine are administered with some of the foregoing drugs to abolish the irregular peristalsis or griping which some of them produce. For the same reason they are useful in spastic constipation.

Strychnine is commonly used for its tonic action on the muscle walls of the intestines: it produces its effect by acting on Auerbach's Plexus.

As noted elsewhere some hormones from ductless glands may be tried where the constipation is occurring with symptoms of a deficiency of that internal secretion, e.g. Thyroid in Hypothyroidism. It is a well proven fact that Pituitary Extract has a markedly stimulating effect on the intestine in post-operative

37.

intestinal paralysis.

Liquid Paraffin, a mixture of liquid hydro-

carbons is unabsorbed from the alimentary canal. It is widely used: in some cases producing an easy motion, but in others it may pass through without producing a motion at all.

The lactic acid bacillus in the form of specially soured milk has been used in the treatment and prevention of auto-intoxication with varying success.

Intestinal Disinfectants. Many have been tried and great results have been claimed from their use, but they are of no specific value.

Enemata. These produce peristalsis mechanically, thermally or chemically. The mechanical stimulation is brought about by the distension of the gut, and may be produced by introducing into the bowel by means of a tube, about two pints of fluid from a container hanging about

eighteen inches above the level of the patient's anus. Fluid introduced in this way flows passively to the coecum before peristalsis is produced.

Fluids at different temperatures may be used as enemata. Cold water (60° F. to 70° F.) has a tonic effect on the musculature of the intestinal tract. Below 60° F. water - or other fluid - may produce excessive stimulation which means violent colic. Warm enemata soothe the bowel by relieving enterospasm.[38.] Gant uses enemata at a temperature of 100° F. to 110° F. in the treatment of enterospasm,[39.] whereas Hurst is of opinion that enemata of a temperature more than a few degrees above body heat injure the intestinal mucous membrane.

Various substances may be added to enemata

to produce chemical stimulation of the bowel. The substances most commonly used are soap, turpentine, salt, glycerine. The action of soap is due to irritation of the mucosa. Turpentine is used where it is desired to expel flatus: similarly salt is used in a "brine enema" (two to four ounces to one pint of water). Glycerine is used by itself in quantities of one drachm to one ounce: the response is generally rapid. Olive oil, four to eight ounces, introduced slowly is soothing and lubricating in its action. In some cases, a high injection of olive oil at night followed by a soap or plain water enema next morning gives most efficient results.

There are various hydro-therapeutic methods which are often of great assistance in the treatment of chronic constipation, if combined with

massage and general exercises. Baths may consist of fresh, salt, or effervescing waters; they are plunge baths, immersion baths, sitz baths, and sweating baths.

Douches and sprays play a great part in this method of treatment.

The treatment of the Sequelae of Chronic Intestinal Stasis if they be severe must be symptomatic.

Hyperacidity must be treated with antacids, hypoacidity with nitro-hydrochloric acid and pepsin if there is poverty of gastric juice - Anaemia will require haematinics.

In cases of prolonged gastric delay, daily lavage of the stomach will assist that organ to recover its tone.

Nervous symptoms must be quietened until

the bowels are got into regular order.

Operative Treatment. In some cases - where tumour, some foreign body, strictures or volvulus is the cause of the non-operation of the bowels - surgical interference may be necessary.

But for Chronic Intestinal Stasis, as a rule, medical treatment suffices. In cases of visceroptosis it has long been debated as to the advisability or otherwise of operation. A small band of surgeons headed by Lane is in favour of immediate operation; they favour colectomy as the only method of cure. The supporters of this theory are becoming fewer and fewer. Another group favour partial colectomy.

But nowadays the general opinion held is that operation is called for only in the event of complete failure of medical treatment. The

operation consists of releasing all kinks by dividing bands and membranes, and, if necessary, covering over raw surfaces with omental grafts.

40.

Lane in his latest discourse on the treatment of Chronic Intestinal Stasis suggests that all operative measures on any portion of the gastro-intestinal tract should be started by an examination of the last kink, and by the careful freeing of the bowel from its acquired attachment to the iliac fossa. This must be done thoroughly and with the greatest care, and when the mesentery has been restored to its normal length and the bowel to its normal mobility, any raw surfaces of mesentery must be accurately covered by peritoneum to obviate the formation of inflammatory adhesions.

Lane states that he has on very many occasions derived the same advantages by this simple

operation as he has previously had from colectomy at a considerably greater risk.

After dealing with the last kink, examine the colon in its entire length, making sure that no secondary constriction has developed, and particularly in the splenic flexure.

V. CONCLUSION.

Chronic Intestinal Stasis has long been known but it is only of recent years that it has been recognised as a definite pathological entity. As our knowledge of the condition advances with newer means of investigation it is now regarded as an all-important step in the production of many and varied morbid processes.

The symptoms may be local or remote - confined

to the abdominal cavity or spread to all organs of the body. The distal symptoms are caused by absorption of toxins from the alimentary tract or, as it is called, auto-intoxication.

The severity of the symptoms varies markedly in different subjects: some suffering severely if defaecation is delayed more than twenty-four hours, while others may not obtain an evacuation of the bowels for several days and yet experience little or no discomfort.

It would appear that auto-intoxication occurs because of some defect in the poison neutralising powers of the liver.

Treatment is almost entirely medical and until that fails completely surgical interference is uncalled for. When the latter is undertaken, colectomy is entirely unnecessary, and the opera-

tion should be confined to dividing adhesions and connecting bands.

In the words of the proverb, "Prevention is better than Cure." Conditions which predispose to Chronic Intestinal Stasis should be eradicated, and from the cradle, the child should be taught to realise the great importance of securing regular evacuations of the bowel contents.

VI. BIBLIOGRAPHY.

1. "The Prolongation of Life", 1907. 67.

2. Med. Press. 1921. April 6. 279.

3. Asher, L. Deutsche Medizinische Wochenschrift, XXXIV. I. 1916.

4. Ott. Proceedings of the Physiological Society: Journal of Physiology, 1904.

5. Weiland, W. Pfluger's Archiv. CXLVII. 171. 1912

6. von Bergmann, G. Zeitschrift fur experimentelle Pathologie und Therapie. XII. 221. 1913.

7. Hurst, A. H. Constipation and Allied Intestinal Disorders. p.63. 1920.

8. Gagenbaur. Elements of Comparative Anatomy. 1878. p.562.

9. "The Prolongation of Life", 1907. 151.

10. Mutch, Nathan. The Operative Treatment of Chron: Intestinal Stasis: by W. Arbuthnot Lane. p.114. 1918.

11. B.M.J. 1924. 15th March. 461.

12. Lane. The Operative Treatment of Chronic Intestinal Stasis. Preface to Fourth Edition IX. 1918.

13. Metchnikoff. The Prolongation of Life (English Translation by F. Chalmers Mitchell. pp. 67-72. 1910.

14. Gant, S. G. Constipation, Obstipation and Intestinal Stasis, p.129. 1916.

15. Lane. "The Operative Treatment of Chronic Intestinal Stasis." 26.

16. Virchow, R. Virchow's Archiv. V.335. 1853.

17. Hurst, A. "Visceroptosis" in "International Clinics", 25th series. IV. p.113. 1915.

18. Hurst, A. Constipation and Allied Intestinal Disorders, p.304. 1919.

19. Surg. Gym. and Obst. 1913. July. 71.

20. Medical Annual: Visceroptosis. 1920. 396.

21. Quart. Journ. Med. 1922. Jan. 144.

22. Quart. Journ. Med. 1921. April. 215.

23. Lane. The Operative Treatment of Chronic Intestinal Stasis. 238.

24. Med. Press. 1921. Oct. 26. 336.

25. Lane. The Operative Treatment of Chronic Intestinal Stasis. 247.

26. Lancet. 1921. ii. 1266.

27. Chapple, H. B.M.J. January 1914.

28. B.M.J. 1920. ii, 959.

29. Lane. The Operative Treatment of Chronic Intestinal Stasis. 66.

30. Hurst, A. Constipation and Allied Intestinal Disorders, p.243. 1919.

31. B.M.J. 1922. i. 941.

32. Mutch, Nathan. The Operative Treatment of Chronic Intestinal Stasis, by W. Arbuthnot Lane. p.267. 1918.

33. Metchnikoff, E. The Prolongation of Life, p.167. 1910.

34. Medical Annual. 1922. 95.

35. Hale White, W. Materia Medica, etc. 1918. p.97.

36. Hurst, A. Constipation and Allied Intestinal Disorders, p.350, 1919.

37. Cushing, A. R. Pharmacology and Therapeutics or the Action of Drugs, p.387. 1918.

38. Gant, S. G. Constipation, Obstipation and Intestinal Stasis --, p.233. 1916.

39. Hurst, A. Constipation and Allied Intestinal Disorders, p.360. 1919.

40. B.M.J. 1924. 26th Jan. 144.

VII. APPENDIX:

containing notes of cases illustrative of condition.

Case No. 1. P.S. - a youth, aged 19, a printer by trade. History of obstinate constipation for the past few years. The period between evacuations had varied, latterly having been as much as a week, in spite of castor oil, senna, salts and other aperients. He had experienced little if any abdominal discomfort up to a week before admission to hospital, when he began to suffer pain down the left side of the abdomen. This pain was somewhat aggravated by the taking of food: there was no heartburn and no sickness: little loss of appetite. His main complaint was headaches, general in distribution.

His last appearance before me was on account of a septic condition of the right heel due to pressure from a tight boot. The skin took an abnormally long time to heal under boric foments. Another period of treatment was for acne.

He stated that his diet consisted of meat, vegetables, puddings and eggs, the latter in fairly large numbers. He has not taken part in games, his only exercise being obtained in his walking to and from his

work: he did not cycle: he drank no alcohol of any sort and was a very moderate smoker.

A few days before admission he was put on a mixture of the liquid extracts of cascara and licquorice and on admission reported that his bowels had acted freely.

On examination he was a well nourished, muscular young fellow with acne, and a muddy, dirty complexion; weight 8st. 1 lb. Temperature 99° O. Gums healthy and teeth in sound condition.

The abdomen moved freely with respiration. Slight tenderness was elicited only along the course of the descending colon. The abdomen was tense and the muscles were held rather rigidly. The faeces were dark brown in colour, and like small pellets. Haemorrhoids were present:

Urine - S.G. 1006: acid: albumen nil; sugar nil.

Circulatory System - Pulse 78: regular in force and rhythm and of good volume. Heart sounds closed.

Nervous system - the patient complained of headaches: otherwise there were no subjective symptoms. Reflexes normal.

Diagnosis: a case of slight chronic intestinal stasis with few toxic symptoms.

X-rays revealed that within 15 minutes the barium showed in the stomach and some small amount

in the small intestine. Four hours after the meal a small quantity of barium was observed in the ileum, the majority being in the coecum and first part of the ascending colon. Eight hours after the meal the barium shadow occupied the coecum, the ascending colon and it was spreading along the transverse colon. Twelve hours after the meal, some barium had been evacuated while some remained in the transverse colon and descending colon. Twenty-four hours after the meal there remained only a small trace of barium in the rectum. It must be pointed out that the Barium meal was given and X-ray photos taken about four days after patient had begun treatment, and consequently the picture shows a different condition than if the examination had been made before treatment was commenced.

The patient was confined to bed for a week as his temperature remained steady at 99.0. He received half an ounce of liquid paraffin by the mouth each night, and six ounces of olive oil injected per rectum with a long tube. This he retained overnight and he was given a saline enema at 70° in the mornings. On the second morning, the wash-out produced a most copious result, also that on the third morning. Within three days the

bowels began to act normally in the afternoons, and the wash outs were discontinued, a further half-ounce of liquid paraffin being given by the mouth each morning. Light diet was given for the first few days on account of the temperature remaining up, and was later increased to a stimulating diet containing a limited quantity of meat, extra quantities of vegetables, porridge, jams, fats, fruit (cooked and uncooked), and abundant fluids. Abdominal exercises were ordered.

All abdominal discomfort rapidly disappeared. Patient began to feel more active; the complexion cleared; the headaches became much less severe; he gained weight; and the bowels were acting regularly and well without artificial aid on his discharge from hospital.

Case No. 2. A.B. - a domestic servant, aged 34. Complained of tiredness, loss of appetite, sickness, "biliousness", headaches. Her National Insurance medical record card shows that she consulted her doctor for "vertigo" in January 1921, "Headaches" in May 1921, "Liver" in March 1922, "Anaemia" in May 1922. She came under my care first in December 1922, having ~~an~~ influenza.

Later, she was under treatment for anaemia for three months, during which I tried various aperient medicines to regulate her bowels, with scant success.

Patient was a poorly nourished woman, of a pale, dirty, muddy complexion; her eyes looked dull and "heavy". The lips and conjunctiivae were pale. She stated that she had taken all manner of aperients but had never been able to get her bowels into regular daily action. She went three and four days between motions. She could eat but little on account of pain following the taking of food. She complained of pain in the right side of the abdomen, and along the course of the descending colon: these pains were increased by aperients.

She had a complete upper and lower artificial denture. The gums were healthy: the tongue coated. On palpation, she complained of tenderness in the coecal region, in the epigastric region, and in the left hypogastric and iliac regions. The descending colon was palpable.

Nervous system: she complained of headaches and insomnia. She was distinctly neurotic and depressed.

Reproductive system: "periods" irregular; she suffered much pain for two or three days preceding them; the "period" was scanty.

Circulatory system: pulse 80: regular: of poor volume: a systolic haemic murmur was heard. Patient was markedly anaemic.

Skin: the skin was cold; perspired freely, the perspiration having a slightly disagreeable odour. Pigmentation was marked under the eyes.

The urinary and respiratory systems presented no abnormalities.

The breasts were nodular in the upper and outer quadrants.

A Barium meal showed a great degree of intestinal stasis as the following report shows. Fifteen minutes after the barium meal the shadow occupied the stomach and a small quantity of barium was making its way through the duodenum. Four hours after meal the stomach was clear: a small amount of barium showed in the ileum while the majority was in the coecum and ascending colon. Eight hours after meal the shadow was still in the coecum and ascending colon. Twelve hours after meal the condition did not seem altered from eight hours. Twenty-four hours after meal the majority of the barium still remained in the ascending colon while a small amount had entered the transverse colon. Thirty hours after meal there still remained a large quantity of barium in the ascending

colon and a trace had reached the splenic flexure. Forty-eight hours after the meal, the ascending colon still presented a shadow of barium, the majority being in the transverse colon, with a trace in the pelvic colon. Fifty eight hours after the meal there was still a slight shadow in the ascending colon, the majority of barium being in transverse and descending colon. In another twenty hours from the last examination, i.e. 70 hours after meal, the barium still persisted in the descending and pelvic colon.

Diagnosis: A case of marked chronic intestinal stasis with many toxic symptoms affecting various systems of the body.

Treatment as for case No. 1 with the addition of abdominal massage and electricity brought about great improvement in this woman's condition. The symptoms of indigestion quickly disappeared. The tongue cleared, the complexion improved, the "heaviness" disappeared from the eyes, the headaches became less severe and gradually disappeared, the appetite improved and the patient appeared "a different creature".

Prints from the X-ray negatives of this case appear at the end.

Case No. 3. J. McN. - a farm labourer aged 59. I was called to see this man one evening in April 1924 and found him lying on his left side in bed, with his knees drawn up. Suffering acute agony in the abdomen and unable to move. He had been brought home from work. His history was that he had been getting more and more constipated of late. He had had a slight evacuation the day before as a result of a dose of castor oil. He had vomited before I saw him, and passed a small quantity of flatus. The hernial openings were free; nothing was felt per rectum. Palpation of the abdomen was useless and one desisted on account of the pain evolved.

The diagnosis was Faecal obstruction and I had him removed to hospital. The treatment carried out there (he was not under my care) was oil by the mouth and per rectum. After the bowels had been thoroughly cleared by these methods, a barium meal showed no real delay.

I afterwards learned from this man that he had had a similar attack some time in 1917, since when he had been quite fit up till the attack of obstruction in 1924.

He has continued well since leaving hospital and the bowels are acting regularly.

Case No. 4. D.C. - a schoolgirl, aged 13. Her mother brought her complaining of pains in the chest, headaches, and general nervousness. The girl had been in a sanatorium about three years ago for Pulmonary Tuberculosis and discharged for observation by the Tuberculosis Officer, who now reported the child free from Tuberculosis.

Patient was a tall (for her age), thin, nervous child: of sallow complexion (but I understand there is a hereditary factor in the swarthy skin). There was a slight degree of anaemia. Her carriage was poor: her head drooped forward, her chest was flat, her shoulders drooped, her abdomen protruded slightly - a rather typical picture of Walton's virginal type of "ptosis habitus". The tongue was furred. Nothing abnormal was felt on palpation of the abdomen, but the child complained of tenderness in the epigastrium. Her mother stated that the bowels were regular.

Nervous system: the child was stated to be restless in her sleep, and very nervous. She complained of headaches (apparently not connected with eyestrain as the eyes had recently been tested and her glasses found to be correct for her error of refraction), and vague pains all over her body.

Circulatory system: heart sounds closed.

Pulse 86: regular.

Urine neutral: S.G. 1018: albumen nil; sugar nil. On one occasion the urine contained a trace of albumen.

Diagnosis: Auto intoxication arising from chronic intestinal stasis. This diagnosis was confirmed by Dr. Theodore Thomson who saw the child for me at the Sick Children's Hospital, Great Ormonde Street.

The patient removed from my district before any result of treatment was seen.

Case No. 5. Baby C - aged 2 years, female. This child presented signs of constipation - crying with pain when put to stool, irritability, loss of appetite, pasty complexion and heaviness of the eyes, with a slight elevation in temperature. The report on the bowels was that they acted very slightly each day. She was put on a rhubarb and soda mixture and enemata of plain water, soap and water and olive oil tried without any improvement. A small glycerine enema was given and this brought down to the anus a scibbolous, clay-coloured mass of faecal material (the size of a large chestnut) which I removed with the aid of a hernia needle. With this faecal obstruction removed the child

rapidly improved and recovered her accustomed brightness: the appetite returned, the eyes cleared and the bowels acted regularly and with ease.

Case No. 6. Mrs. E - aged 53: a hard-working woman of small stature: complained of "indigestion" and headaches. The mother of eight children. She stated that the bowels were "regular". She presents a typical picture of Walton's maternal type of "Ptosis Habitus" - with the thorax unaltered, but the lumbar curve lost, and the loose, baggy and sagging abdominal wall due to overstretching of the muscles in repeated pregnancies. On palpation, tenderness was elicited along the course of the entire colon. Examination of the rectum proved negative.

Nervous system: the neurasthenic element was most marked. Knee jerks and other reflexes were exaggerated. She refused to be X-rayed or to go into hospital.

Reproductive system: the menses still continue although they are now rather irregular.

The circulatory and respiratory systems presented no abnormalities.

Urine: acid: S.G. 1015: albumen nil; sugar nil.

The diagnosis was Visceroptosis.

The treatment recommended here was a belt of the Curtis type which produced a certain amount of improvement: the bowels becoming certainly more regular being assisted by a Senna mixture, and the neurotic symptoms becoming less marked.

Case No. 7. B.S. - a girl, aged 20: gave a history of constipation from childhood. The periods between motions had become progressively longer - from every second or third day until the bowels opened only once in about eight or nine days. Aperients had proved of little help in relieving the condition, as had a special diet including plenty of vegetables and fruit.

She complained of loss of appetite and a feeling of fulness after meals, a considerable amount of flatulence, and occasional attacks of colic.

She had been taking liquid paraffin for about a month before admission, but the result had been poor.

The patient was a well developed girl of average stature, somewhat anaemic with a muddy complexion.

Gums and teeth were healthy. Tongue furred.

The abdominal wall contained abundant subcutaneous

fat. The colon was palpable along most of its course. The rectum contained impacted masses of faeces.

Nervous system: The neurasthenic element was pronounced. She complained of headaches - mainly frontal - insomnia, coldness and numbness of the hands and feet.

Circulatory system presented no abnormalities

Reproductive system. Here the menses were irregular, there was considerable pain for about 24 hours preceding the period and the loss was very small.

The urinary system showed no subjective or objecting symptoms.

A Barium meal showed stasis in the colon, most marked in the coecum and ascending colon: the diagnosis being intestinal stasis.

General improvement followed a course of enemata - high olive oil injections retained overnight, followed by saline enemata at 65° - 70° F. in the mornings for one week; castor oil and olive oil by the mouth at nights; a diet consisting of plenty of green vegetables and fruit; abdominal massage and exercises. The bowel gradually recovered tone, the aperient was gradually reduced and the wash-outs discontinued: the bowels moving

regularly each motning. The neurasthenic symptomd decreased, the headaches were less frequent and less severe, the appetite improved, the complexion cleared, and the coldness of the extremities had in great measure disappeared.

Case No. 2. 4 hours after Barium meal.

Case. No. 2. 12 hours after Barium Meal.